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CLINICAL NOTES

STRANGULATION OF A DIAPHRAGMATIC HERNIA AS A COMPLICATION OF ELECTRIC SHOCK THERAPY

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The post-electric shock complication to be described is unmentioned in the popular textbooks (1, 2, 3) on the subject of electric shock, and it appears uncommon enough to warrant mention.

The patient, a 72-year-old woman, was referred to the hospital because of a serious depression accompanied by agitation, loss of appetite, insomnia, and marked loss of interest in life. There were no findings of organicity. Electroshock therapy was recommended and instituted.

An x-ray diagnosis of diaphragmatic hernia had been made some months previous to her admission. At High Point Hospital physical examination and laboratory procedures were negative. The hospital's internist¹ reported her to be especially well preserved for her age.

The patient received no premedication and cooperated fully for her treatment. She was subjected to a grand mal convulsion by the passage of 130 volts for 0.5 seconds, with glissando. After the seizure there followed an unusually long period of apnea. When respirations were resumed they were weak and laborious, as if a deep breath could not be drawn.

Her reaction was unusual enough to produce alarm and an air-way was therefore introduced on the assumption that the pronounced apnea and cyanosis were due to a collapse of the tongue. While the air-way was inserted it was noted that the patient's jaw was very flaccid. Some slight improvement in respiration followed, but was short-lived.

The patient became more cyanotic, began perspiring, and most alarming of all, she remained inert. Her only muscular activity was a gasping attempt at breathing. Artificial respiration and 2 c.c. of coramine injected subcutaneously were of no help. Her condition became worse and her color turned

quite pale. A vein was finally found, permitting the injection of 7 c.c. of coramine. Shortly thereafter she began to breathe regularly, and soon became conscious. Her pulse was regular at 84 per minute and beat at this rate in the subsequent hours.

On awakening she complained of marked pain in the upper epigastrium, became nauseated, and actually vomited. The pain disappeared within the first 2 hours, but the unusually pronounced nausea persisted all day, despite appropriate medication.

Within 30 minutes after the treatment, our internist examined the patient. He had been unaware of the history of diaphragmatic hernia but suggested that "a diaphragmatic hernia could possibly have become strangulated in the course of the shock therapy in order to produce all of her symptomatology." He permitted the patient out of bed after recovery from its initial ill-effects.

In the light of the history and x-ray findings (see plate), the nature of the initial respiratory distress, unrelieved by the ordinary procedures usually successful, would suggest a diaphragmatic herniation as the cause. Presumably the contraction of the abdominal walls was strong enough to push a portion of the stomach through the hernia. Tonic contraction of the diaphragm may very well have followed. Only the intravenous injection of a respiratory stimulant seemed to re-establish normal diaphragmatic action.

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CONCERNING THEORIES OF INDOLES IN SCHIZOPHRENIGENESIS¹

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Recently considerable interest has been aroused in several theories which would at-

tribute to a biochemical disturbance a significant role in the development and maintenance of schizophrenic reactions. Prominent among these is what might be called the indole theory, of which one statement is that

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proposed by Hoffer, Osmond, and Smythies (1) of the existence of an "M" (mescaline-like) substance either containing an indole nucleus or from which an indole nucleus could be derived. In addition to the 6 substances referred to by these authors, namely mescaline, lysergic acid diethylamide (LSD-25), harmine, ibogaine, hashish, and adrenochrome, a seventh indole, bufotenine, has received attention. The implication is that these substances are capable of producing a hallucinatory state without disorientation and of a schizophrenic rather than a delirious character.

Concerning mescaline and LSD so much of the literature is readily available that we need make no comment, except to remark that in Europe more than in the United States these compounds are looked upon as deliriants.

Hashish does not belong among the indoles at all, for the active principles are nonalkaloidal cannabinoids.

The situation with harmine is extremely complicated. A careful review of the literature of the past 100 years reveals no record of pure harmine or its related alkaloids producing visual or auditory hallucinations. The few statements to the contrary all seem to be on hearsay evidence and, in any event, do not exclude delirium. Gunn (2) studied harmine and a number of its derivatives over a period of 25 years. He stated in a recent letter that he has never observed any psychic effects from these compounds. Others (3) noted as the only psychic effect a sense of well-being. Attribution of hallucinogenic properties to harmine is evidently due to the fact that it is found in various species of *Banisteria* and *Haemadictyon* in South America. Decoctions of these plants produce hallucinations of a delirious nature in the presence of severe intoxication (4). Lewin (5) was able to obtain response of crude *Banisteria* in his laboratory but could not obtain it with pure harmine. His famous work with mescal (*Anhalonium lewinii*) makes his observations particularly valuable.

Concerning ibogaine, the natives of French West Africa do not ascribe to it any hallucinogenic property. "When questioned they insist that it has an action identical with that

of alcohol without impairing the reason" (6). The only reference to hallucinogenic properties is in a paper by Lambert and Heckel (7) in which they assume hallucinations in a dog from its behavior.

As to adrenochrome, not only has the supposed action been denied by Rinkel (8) but a later paper by the Saskatchewan group tacitly negates the original assertion (9).

Finally, concerning bufotenine, we learn that in Haiti a snuff, *cohoba*, has been used as an intoxicant. Bufotenine constitutes 1% of the weight of the seed (10). Dr. Jacques Fourcand investigated the use of *cohoba* by the Haitian natives for us in June 1955 and reported that, following insufflation, a period of rigidity and staring is followed by convulsions. In the postconvulsive excitement and subsequent stupor there are visual and auditory hallucinations, evidently therefore in a delirious state.

On the basis, therefore, of what we have been able to ascertain, the concept that schizophrenia is related to a disturbance of indole metabolism does not receive support from the actions of hashish, harmine, ibogaine, adrenochrome, or of bufotenine.

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